

## Mutation rate and survival with ICR-170

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# Mutation rate and survival with ICR-170

## **Abstract**

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Radford, A. Mutation rate and survival with ICR-170.

The mutation system of Reissig  
(1963 J. Gen. Microbiol. 30:317)

in which pyr-3 forward mutations locking aspartic transcarbamylase but retaining carbamyl phosphate synthetase activity are selected, is one of the few systems in which forward mutation rates can be accurately and directly measured. This system was used to investigate the mutagenic activity of ICR-170 (generously donated by H. J. Creech ).

Conidia of the arg-2 (33442) strain were grown on synthetic cross medium supplemented with sucrose and arginine and harvested at 6-9 days. ICR-170 treatment was carried out in pH 7.0 phosphate buffer, and the reaction was stopped by transferring the conidia to pH 8 buffer. The treated conidia were overplated on petri dishes of sorbose medium at a concentration of  $1-2 \times 10^6$  conidia per petri dish. Eighteen hours after plating, a third layer containing lysine and canavanine was added to the petri dishes to reduce the residual leaky growth of the unmutated arg-2 conidia. All steps involving ICR-170 were carried out in red light to prevent the occurrence of photodynamic mutation, and as an added precaution the plates were kept in darkness or red light for 24 hrs after treatment. The plates were scored after incubation at 25°C for seven days.

Compared to the data on the related compound acridine yellow (Reissig 1964 Neurospora Newsl. 6: 16), there is no doubt that ICR-170 is an effective mutagen. The differences between the concentration-dependent curve (Figure 1a) in which mutation rate and kill increase linearly with dose, and the time-dependent curve (Figure 1b), show that ICR-170 is very rapidly taken up into the conidia, and has its maximum effect within the first few minutes of exposure. The mutants induced in this experiment are currently being investigated to determine their nature. This work was supported by NIH Grant No. AI-01462. ■ ■ ■ Department of Biological Sciences, Stanford University, Stanford, California 94305.

FIGURE 1a

SURVIVAL AND ARG-2 SUPPRESSION

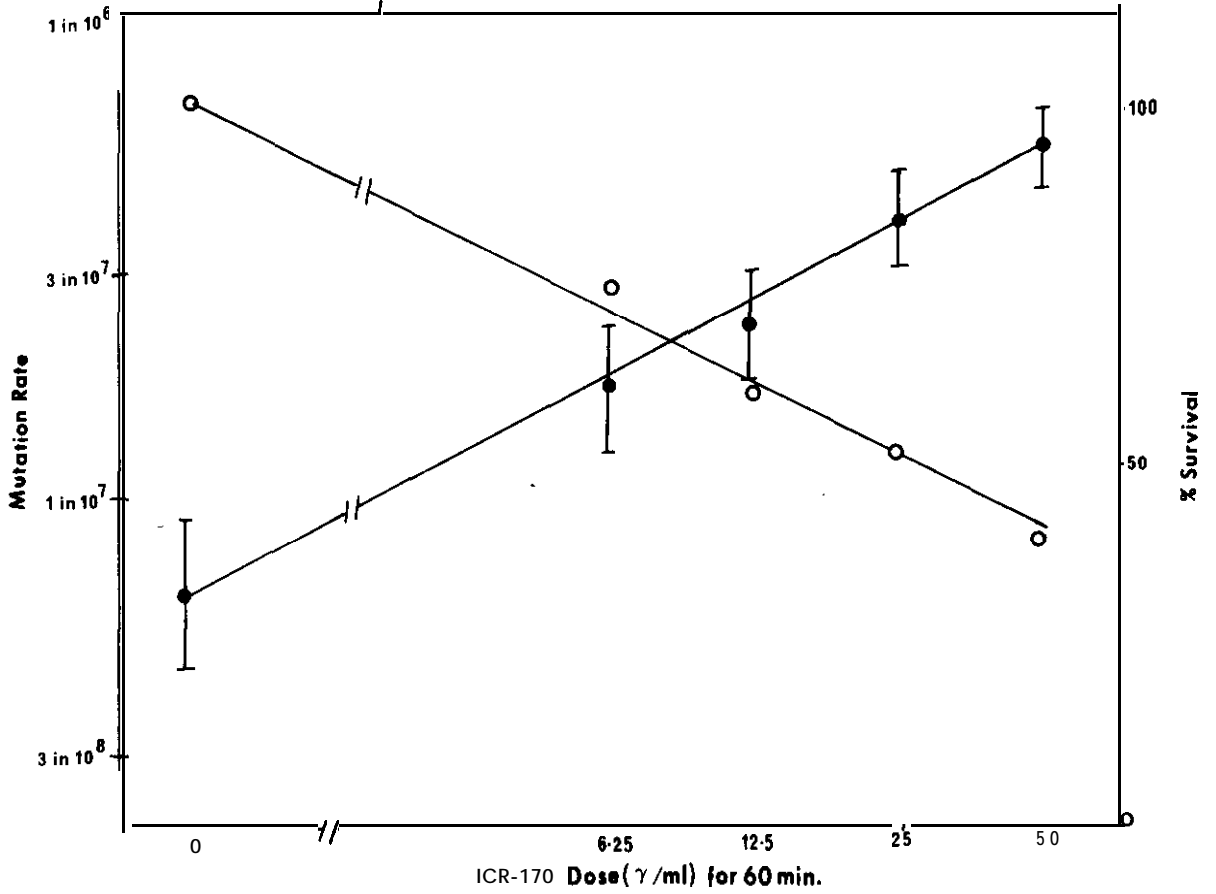


FIGURE 1b

